MEDICAL PRACTICE

Scientific Basis of Clinical Practice

Practical Effects of Blood Group Incompatibility between Mother and Fetus

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It was Ford[1] who first suggested that advantages and disadvantages, such as susceptibility to certain diseases, would be found to be associated with the human blood groups. Nevertheless, it was many years before practical work was started on the subject and only when Aird *et al.*[2] had shown that a significantly high proportion of patients with cancer of the stomach belong to group A and of those with peptic ulcer to group O did the idea become widely accepted. Though of great theoretical interest, such associations between blood groups and disease are not of help to individual patients, and the purpose of this paper is to draw attention to other situations which do have medical (or veterinary) implications in particular cases and yet which also play a part in maintaining the polymorphisms. Such, for instance, are disorders due to interactions between the blood groups of mother and fetus which may result in the various types of haemolytic disease of the newborn, and, though this is more speculative, where blood group compatibility may influence the course of certain types of cancer.

The keynote of haemolytic disease of the newborn, whether it be due to ABO or Rh incompatibility, is how *in*frequently the fetus is affected compared with the chances of how often it might be. This is because there are several naturally occurring protective mechanisms, and it was one of these which led to a successful method of preventing rhesus haemolytic disease.

Blood Group Incompatibilities Affecting the Red Cells

RELATION TO EARLY ABORTIONS

It is now possible to group early abortuses, and there seems no doubt that ABO incompatibility between these and their mothers is a factor in causing the abortion. This had been suspected for some time from a consideration of the frequency of abortion in couples where there was ABO incompatibility, but this does not necessarily mean that the fetus would be incompatible. For example, a group O mother married to a group A man may easily have an O baby, as the father will often be heterozygous AO. In a recent study where the abortuses were grouped, Takano and Miller[3] calculated the expected frequencies of ABO incompatible conceptuses, assuming that the blood type distribution among the fathers was the same as that in the general population and that there was no prezygotic selection. The total number of incompatible abortuses observed was significantly greater than expected ($P < 0{\cdot}01$). In other words, the incompatible conceptuses were more likely to be aborted than the compatible ones.

ABO HAEMOLYTIC DISEASE OF THE NEWBORN

This is usually a mild disease characterized by haemolytic jaundice which clears up spontaneously. It is clinically apparent in only about 3% of newborn infants, whereas in about 20% of births there is mother/child ABO incompatibility. There must, therefore, be protective mechanisms, and some are well recognized, but many problems remain, not the least being that there is no agreement as to what actually constitutes the disease. Some facts are as follows.

(1) Mothers of group O are almost the only ones concerned, and in contrast to rhesus haemolytic disease (see below) it is often the first ABO incompatible child which shows signs of the disease. The reason for group O mothers usually being involved is that they form 7*S* anti-A or anti-B more readily than group A or group B mothers (and some of their "naturally occurring" anti-A and anti-B may be of this type) though why this is so is unknown. The fact that the first baby is often affected may sometimes be explained by the mother having been immunized by some other (non-human) A-specific antigen—for instance, by an earlier in-

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jection containing horse serum. The formation by the mother of 7*S* antibody is very important, since only this will cross the placenta and therefore damage the baby; the naturally occurring anti-A or anti-B in women who are not group O is almost all of the 19*S* type, which having a larger molecule remains in the maternal circulation.

(2) In contrast to rhesus haemolytic disease of the newborn (where all clinically affected babies are Coombs-positive) there is no certain diagnostic test for ABO haemolytic disease. In ABO disease badly affected children may not show a positive Coombs test and in some cases where this test is positive the children are not affected at all. The reason for this is not known, but possibly a subtype of 7*S* antibody is involved requiring special techniques for its demonstration.

(3) Babies who are ABH secretors are more likely than non-secretors to cause a rise in titre of 7*S* immune anti-A or anti-B, the blood group substance being known to cross the placenta at delivery and possibly earlier. A secretor baby can, therefore, act as an immunizing pregnancy.

(4) The fetus has a variable amount of blood group substance in its plasma, and the more there is present the more likely is any 7*S* maternal antibody to be neutralized, thereby preventing it from damaging the fetal red cells.

(5) The antigens on the fetal red cells are said to be poorly developed in some cases and therefore may not immunize normally, but how far this is really true is doubtful since there never seems any difficulty in grouping a baby from the cord blood. If the antigenicity is poor it raises difficulties in relation to protection against Rh immunization by ABO incompatibility.

RH HAEMOLYTIC DISEASE

This is a much more serious condition, the babies often requiring exchange or intrauterine transfusion, and the disease is also the cause of a proportion of stillbirths and brain damage. It is, therefore, a subject of considerable anxiety to many parents. Nevertheless, despite the fact that 15% of the population lack the Rh factor, only a small proportion of such women who bear Rh positive babies actually form antibodies. Again, therefore, protective mechanisms have to be considered, and there are many factors which influence Rh immunization. Some of these are now discussed.

ABO Incompatibility between Mother and Fetus

It has long been known that incompatibility between mother and baby (for example, mother: Rh-negative, group O; fetus: Rh-positive, group A) protects against Rh immunization. Levine[4] was the first to recognize this when he studied the ABO and Rh groups of the parents of erythroblastotic infants and observed an excess of matings in which the father's ABO blood group was compatible with the mother's. Nevanlinna and Vainio[5] made a great contribution by studying families in which the mother made anti-D even though the father was ABO incompatible (in this case a baby could, of course, be compatible if the father was heterozygous). They concluded that an ABO compatible fetus is much more likely to immunize its mother than an incompatible one. Clarke *et al.*[6] confirmed this in an investigation of 91 families, in all of which Rh haemolytic disease had occurred. No fewer than 23 of these families were from ABO incompatible matings as between mother and father and in 14 of them the immunizing fetus could be determined with certainty. They found that its ABO group was always compatible with that of the mother, the probability of this occurring by chance being less than 1 in 500. Most people think that the protection is restricted to pre-immunization (affected children being equally commonly ABO compatible and incompatible), but some authorities believe that a minor degree of protection may be conferred on an ABO incompatible Rh positive fetus even after the mother has been immunized to Rh.

Experimental confirmation of protection by ABO incompatibility has come from Stern *et al.*,[7] who gave Rh positive blood to Rh negative subjects; when the injected cells were ABO compatible, 17 out of 24 formed anti-D while injection of ABO incompatible cells resulted in only 5 out of 32 being immunized. It is interesting that the protection is far more effective when the mother is group O than when she is A or B, and this is probably because such mothers are more likely to have both 7*S* and 19*S* types of A (or B) antibody.

The mechanism by which ABO incompatibility protects against Rhesus immunization is uncertain.

Race and Sanger[8] suggest that incompatible cells would be rapidly destroyed and thus unable to provide an antigenic stimulus. The destruction might be either intravascular or extravascular, the latter occurring predominantly in the liver[10] and fetal cells sequestrated there, rather than in the spleen, are probably less well placed for antibody stimulation. The other explanation is that of clonal competition for antigen. Here it is postulated that a group O woman, having many anti-A or anti-B forming cells available, would take up A or B erythrocytes and therefore little Rh antigen would reach the few anti-Rh forming cells. Against this, however, is the fact that in these circumstances the anti-A or anti-B titre does not usually rise.

Whatever the truth, protection by ABO incompatibility appears to be non-specific—that is, antibody formation against all the antigenic determinants on the erythrocytes is suppressed, a point of great importance when considering the mechanism by which prophylactic anti-D gammaglobulin exerts its effect.

A study of the figures in Table I shows that many fewer fetal cells remain in the maternal circulation when there is feto-maternal ABO incompatibility.

TABLE I—*Distribution of Fetal Cell Scores after Delivery. ABO Compatible and ABO Incompatible first Pregnancies (Liverpool)*[9]

Pregnancy		Fetal cell score*							Total
		0	1	2	3–4	5–10	11–39	40+	
ABO compatible	Number	880	411	187	153	150	144	75	2,000
	Per cent.	44·0	20·5	9·4	7·6	7·5	7·2	3·8	100
ABO incompatible	Number	314	65	24	6	5	3	0	417
	Per cent.	75·3	15·6	5·8	1·4	1·2	0·7	0	100

*A score of 5 suggests approximately 0·2 ml fetal blood in the maternal circulation. Reproduced by courtesy of the author and Messrs. Munksgaard.

Rh Immunization in Relation to Type of ABO Compatibility

It is clear that mother and child may be of the same ABO group (for example, mother O, baby O), or different ones (for example, mother A, baby O) though in both cases the baby is ABO compatible, its cells not being agglutinated by maternal serum. Renkonen *et al.*[11] found that immunization was more likely in cases where the blood groups of the mother and of the immunizing fetus were identical. Why this is so is unknown, but it is mentioned here because of a similar ABO finding in choriocarcinoma.

Rh Genotype of the Baby

The number of D antigen sites on cells of different Rh types was estimated by Rochna and Hughes-Jones[12] using purified ^{125}I-labelled anti-D IgG. Their findings agreed with the serological observations that cDE cells react more strongly with antibody in vitro than those with CDe and that on the whole homozygous cDE/cDE cells are agglutinated more strongly than are those of any other Rh type. Murray[13] studied the Rh types of children whose mothers had become immunized and concluded that a cDE/cde fetus was more likely to immunize than a CDe/cde one. Also, it was shown later that in first-affected infants those of cDE/cde were more likely to need treatment for haemolytic disease.

Sex of the Baby

Renkonen and Timonen[14] published data which suggested that Rh negative mothers are more likely to be immunized by male than by female fetuses. The sex ratios of the immunizing fetuses in their two series of cases were 1·44 and 1·74, respectively. Support has come from Woodrow and Donohoe[15] in Liverpool, for they reported that in 63 first pregnancies immunized six months after delivery the sex ratio was 1·5—but the reason is unknown. Since immunization is caused by feto-maternal haemorrhage one would have expected larger bleeds where the fetus was male, but in fact Woodrow and Donohoe found the distribution of the post-delivery fetal cell scores to be the same in both sexes.

Other Genetic Factors

The variation of the immune response to the D antigen both by male volunteers injected with Rh-positive blood and by women after pregnancy may be because the ability to make antibodies is in part inherited. If so, one would expect Rh haemolytic disease to run in families, and this might be tested by investigating Rh-negative sisters of immunized women and comparing the immunization rate with that in sisters of non-immunized controls.

Size of Transplacental Haemorrhage (TPH)

A survey of Rh-negative primiparae was carried out in Liverpool and 760 of these women who had had Rh-positive, ABO compatible babies were tested for fetal cells soon after delivery by the method of Kleihauer *et al.*[16] (Fig. 1), and they were also investigated for Rh antibodies six months later.[15] Table II shows that there is strong correlation between the size of the transplacental haemorrhage and the chance of Rh immunization—the larger the bleed the greater the likelihood.

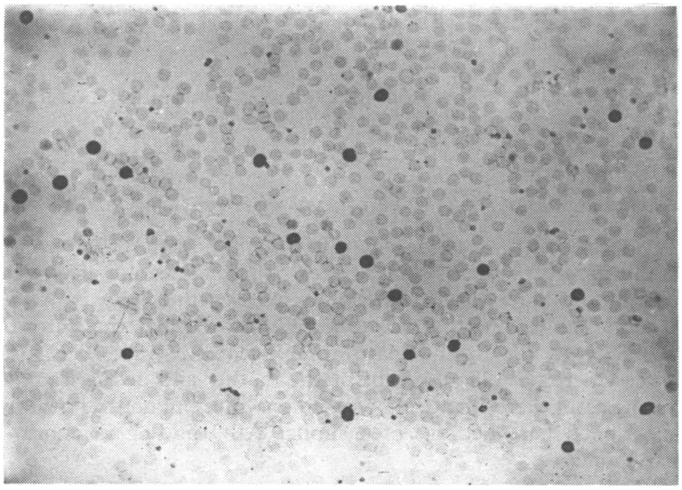

FIG. 1—Fetal cells in the maternal circulation. The fetal cells retain their fetal haemoglobin whereas those of the mother appear as "ghosts", the adult haemoglobin having been eluted from them (technique of Kleihauer, Braun, and Betke.[16])

Nevertheless, this is not all, because as many as 13 of 61 immunized women had had no fetal cells found at delivery, and possibly there are two mechanisms involved in antibody formation. In the first a large transplacental haemorrhage either immunizes at six months or not at all—whereas in the second very small TPHs may result only in "sensibilization" following delivery, and the stimulus of a second pregnancy is needed to produce overt antibodies.

TABLE II—*Relation of Fetal Cell Score after Delivery to Rh Antibody Formation Six Months Postpartum (Liverpool)*[9]

Antibody test six months after delivery	Fetal Cell Score after Delivery							
	0	1	2	3–4	5–10	11–39	40+	Total
Anti-D present	15 (4)	7 (3)	9 (2)	12 (4)	12 (2)	10 (1)	4	69 (16)
Anti-D absent	442	191	88	62	47	36	14	880
Total	457	198	97	74	59	46	18	949
Per cent with antibodies	3·3	3·5	9·3	16·2	20·3	21·7	22·2	7·3

Primiparae. All pregnancies Rh-positive and ABO compatible.
Papain antibodies in brackets (included in large number).
A score of 5 implies approximately 0·2 ml fetal blood.
(By courtesy of the author and Messrs. Munksgaard.)

Immunological Tolerance

A possible reason for an Rh-negative woman not becoming immunized by the red cells of her Rh-positive baby is that she may be tolerant of fetal antigens.

Jakobowicz *et al.*[17] measured the strength of anti-A in the serum of 230 group O and group B recruits both before and after anti-tetanus injections (which contain A specificity). The average rise in antibody was significantly less if the recruit's mother was group A than if she was group O or B.

Theoretically, an Rh-positive woman might make her female Rh-negative fetus tolerant to the Rh antigen, and thus less likely to have an immune response to it in pregnancy than an Rh-negative woman whose mother had been Rh-negative. Race and Sanger[8] give an account of this attractive "grandmother" hypothesis (originally put forward by Rogers Brambell), but in several series of grandmothers' bloods no support for it has been found.[18 19] However, the actual Rh genotypes concerned have not been considered, and this might be important. On the other hand, if, as now seems possible, the fetus can make antibodies, possibly occasional Rh-negative babies get immunized by a transplacental haemorrhage (in the reverse direction) from their mothers, and this might account for cases of immunization or priming where there is no history of previous abortion or blood transfusion.

Immunological Inertia During Pregnancy

Occasionally transplacental haemorrhage takes place during pregnancy, sometimes in the second trimester, leaving plenty of time for antibodies to develop before the end of that pregnancy. In fact, however, it is very rare to find anti-D in primiparae at delivery. Nevanlinna and Vainio[20] found only 4 in 4,153 of such cases and Woodrow and Donohoe[15] only 6 in 2,000, but Chown gives a higher incidence, about 2%.[21]

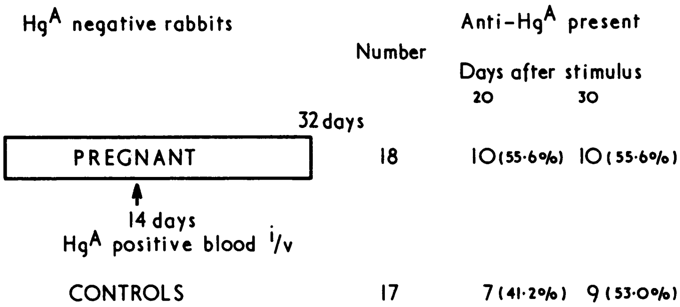

FIG. 2—This shows that Hg^A negative rabbits, when injected with Hg^A positive cells during pregnancy, make anti-Hg^A just as readily as non-pregnant controls. (Reproduced by courtesy of J. C. Woodrow).

To explain this lack of immunization by fetal bleeding during pregnancy, possibly there is a state of maternal anergy during this time, but if so it only applies to primary immunization because women primed by one Rh-positive pregnancy can rapidly develop antibodies quite early in a second one. In Liverpool Woodrow *et al.*[22] have studied the problem in animals and shown conclusively that pregnant rabbits can make humoral antibodies as well as non-pregnant ones (Fig. 1).

PREVENTION OF RH IMMUNIZATION

As has been pointed out above, when there is ABO incompatibility between mother and baby the mother is very unlikely to become immunized to Rh. However, 80% of pregnancies concern ABO *compatibility*, and we thought that we might be able to prevent Rh immunization here by injecting after delivery anti-D antibody. This would, we hoped, render non-immunogenic any Rh-positive cells which had crossed the placental barrier, and it was Finn[23] who first put forward this idea.

After five years of experimental work on Rh-negative male volunteers and postmenopausal women, we showed that it was possible to prevent most cases of immunization and we then designed two clinical trials to test protection in Rh-negative mothers.

First Liverpool Group Trial

This began in 1964 and ended in 1968 except for the follow-up of the subsequent pregnancies.[22 24] The figures are given in Tables III and IV.

TABLE III—*Liverpool Group 5 ml High-risk Trial (Rh Antibodies six Months Postdelivery)*

Centre	No.	Not Immunized	Immunized
Controls			
Liverpool	92 (40*)	72 (30)	20 (10)
Baltimore	31 (14)	23 (10)	8 (4)
Sheffield	41 (15)	31 (10)	10 (5)
Leeds and Bradford	12 (9)	12 (9)	0 (0)
Totals	176 (78)	138 (59)	38 (19)
Treated			
Liverpool	94 (40)	93 (40)	1 (0)
Baltimore	26 (13)	26 (13)	0 (0)
Sheffield	39 (14)	39 (14)	0 (0)
Leeds and Bradford	14 (11)	14 (11)	0 (0)
Totals	173 (78)	172 (78)	1 (0)

*The numbers in parentheses are those when the trial was published.[24] They are included in the larger figures.

TABLE IV—*Liverpool Group 5 ml Trial. (Rh Antibodies at the end of Second Rh-positive Pregnancy)*

Centre	No.	Not Immunized	Immunized
Controls			
Liverpool	38	30	8 (7)*
Baltimore	4	3	1
Sheffield	19	10	9 (5)
Leeds and Bradford	4	2	2
Totals	65	45	20 (12)
Treated			
Liverpool	59	57	2 (1)
Baltimore	12	12	0
Sheffield	10	10	0
Leeds and Bradford	7	7	0
Totals	88	86	2 (1)

*The numbers in parenthesis are mothers who had developed antibodies after the first pregnancy. They are included in the larger number.

The trial was designed to give the greatest quantity of information on the efficacy of the treatment while treating as few women as possible, and each centre adopted the same protocol. Since the likelihood of immunization was found to be related to the size of the transplacental haemorrhage at delivery (see Table II), only previously untransfused primiparae who had had a transplacental haemorrhage with a Kleihauer score of 5 or over (Fig. 1) and who had had an Rh-positive, ABO compatible baby were included (these were presumed high-risk cases) and alternative cases were kept as controls. The dose was 5 ml of anti-D gammaglobulin containing about 1,000 μg of anti-D, given intramuscularly within 48 hours of delivery. The gammaglobulin was prepared by Dr. W. d'A. Maycock and his staff at the Lister Institute, London.

The results were successful (Tables III and IV), there being only two failures in the series. The trial showed that we had, in fact, picked a high-risk group (immunization rate in the controls 22%) and that here the subsequent pregnancies are protected (Table IV). The fact that the subsequent babies are normal is the real proof of the success of the treatment, since "no antibodies at six months" might simply mean that overt immunization had been postponed.

Second Liverpool Trial

Here the dose of anti-D gammaglobulin was 1 ml (about 200 μg), other centres were not involved, and the injection was given to primiparae with a fetal cell score of from 0 to 4 in 50 low power fields.[22]

Table V shows that the six months' figures are again very satisfactory, but in the second pregnancies (Table VI) there are three failures in the treated as against five new immunizations in the controls. This appears paradoxical compared with the first trial but probably these failures are women who had been "primed" —that is, immunized by a bleed early in the first pregnancy but with no demonstable antibodies, their hidden antibody having removed the fetal cells which had crossed the placenta during the pregnancy. If this is so, then the anti-D gammaglobulin would have been given too late.

TABLE V—*Liverpool 1 ml Trial. (Rh Antibodies six Months Postdelivery)*

	No.	Not Immunized	Immunized
Controls	362	349	13
Treated	353	353	0
Totals	715	702	13

TABLE VI—*Liverpool 1 ml Trial. (Rh Antibodies at the end of Second Rh-positive Pregnancy)*

	No.	Not Immunized	Immunized
Controls	127	114	13 (4)*
Treated	128	125	3
Totals	255	239	16 (4)

*The number in parenthesis refers to mothers who had developed antibodies after the first pregnancy. They are included in the larger number.

That priming can occur is well demonstrated by a case of our own. A 70-year-old Rh-negative volunteer had no demonstrable antibodies by routine tests and was injected with 5ml Rh-positive fetal blood. Twenty-four hours later there were many fetal cells in his blood film, but at 48 hours these had all disappeared and anti-D was demonstrable. It was then found that he had had a blood transfusion during the first world war, which had clearly primed him. Had he been a woman and 50 years younger he might well have been considered a case for treatment and would have been classed as a failure six months later.

Other Centres

In the past few years prophylactic anti-D has been administered in most parts of the world where rhesus haemolytic disease is a problem. The results are remarkably uniform, with a failure rate of about 0·5% at 6 months and a further 0·5 to 1% at the end of a subsequent Rh-positive pregnancy. These figures have to be compared with a figure of 8·5% at 6 months and a further 8·5% at the end of a subsequent Rh-positive pregnancy if nothing is done. Most of the failures are probably due to "priming" rather than to inadequate amounts of anti-D.

Haemolytic Disease of the Newborn Foal

This disorder has many parallels with rhesus haemolytic disease. Thus the first foal is rarely affected and immunization is thought to occur by transplacental passage of cells at delivery. Unfortunately, however, the erythrocytes of the foal are mature at birth and therefore it is not possible to score fetal cells in the maternal circulation. Also in contrast to man, the immune antibody made against the foal's incompatible antigens is usually of the aggultination (19*S*) type,[25] and this may be why it does not cross the placenta—though it is secreted in the colostrum, where for a short time it is very toxic. A foal normal at birth quickly becomes ill on suckling and may be dead within 36 hours. Since after this time the milk contains no more antibody, the disorder can be effectively prevented by weaning,[26] and this being so it is perplexing to read that the disease is of considerable economic importance in the bloodstock world and that new methods of exchange transfusion are being devised.[27] The reason may be that weaning, though theoretically easy, is difficult in practice (since a little colostrum may be taken) or it may even not be known that the foal is at risk. Also, a foster mother may be difficult to come by, and colostrum seems to be necessary to ensure that the foal thrives.

By analogy with man the disease ought to be preventable by giving the mare gammaglobulin against the incompatible antigens, but bloodstock breeders are very averse to any suggestion being made that there is an inherited disease in their animals.

Haemolytic Disease of the Newborn Piglet

Here, too, the antibody is transmitted to the offspring in the colostrum.[25] The sow can be immunized by certain fetal red cell antigens during pregnancy (though the pig A antigen cannot be responsible since it is formed only after birth), and also (formerly) by the administration of swine fever vaccine, which was made from pooled pigs' blood. The disease has been induced experimentally by the injection of the sow during pregnancy with repeated doses of the incompatible (heterozygous) male's blood. The incompatible piglets in the litter were all killed by HDN, while those which were compatible were unaffected.

Immunological Factors in Choriocarcinoma

A stimulating though unproven suggestion regarding cancer is that most of us at some time in our lives develop neoplasia but that usually we eliminate the malignant cells, possibly by a mechanism similar to that of graft rejection. Modest support for the hypothesis is afforded by the observation that women with a history of allergy may be less at risk for subsequent cancer than controls.[28] Nevertheless, the latter survey was retrospective and the sex difference remains unexplained. More certain is the fact that patients with renal transplants appear prone to certain forms of cancer (lymphoma and lymphosarcoma) probably owing to prolonged immunosuppressive therapy. Conversely, Mathé[29] has shown that remissions in acute leukaemia can be greatly prolonged if immunity is stimulated by repeated scarification of the skin by B.C.G., though this needs confirmation.

A more profitable approach is in choriocarcinomata. These malignant tumours are derived from fetal tissue and, therefore, one of the chromosomes of each pair is of paternal origin and the growth must be considered as a partially allogeneic graft. This might have some relation to the progress or halting of the disease, which as is well known, runs a very unpredicatble course if untreated. *A priori*, it would seem reasonable that where the mother and the cancer had similar histocompatibility genes the outcome would be worse, and, conversely, where the blood groups on the cancer cells were incompatible it might be kept in check or rejected. There is some support for these views.

Scott[30 31] found that the maternal ABO blood group distribution in women with the disease showed a shift away from O to A, B, and AB—which was in keeping with the hypothesis that feto-maternal compatibility is an aetiological factor, and furthermore the growth is said to be more common in inbreeding communities..[32 33]

Ben-Hur *et al.*[34] found that in three patients with choriocarcinoma skin homografts from the husband were not rejected, whereas those from other donors were. The authors suggest that since hydatidiform moles undergo transformation to choriocarcinomata all women with moles should be grafted with their husbands' skin. When this homograft is not rejected they advise against further pregnancies (one supposes for about two years) since the risk of the development of choriocarcinoma would be high and pregnancy would confuse the diagnosis.

The most recent work on the matter concerns both the ABO and the HLA groups. Bagshawe *et al.*[35] reviewed the ABO blood group data in 260 cases of choriocarcinoma, and showed that the risk of the disease developing after any form of pregnancy is critically related to the ABO groups of both the woman and her husband. Women of group A married to men of group O seem to have the highest risk, whereas women of group A married to men of group A have the lowest: the relative risk of these two groups was found to be 10·4/1. The spontaneous regression of trophoblast after evacuation of hydatidiform mole happened most commonly in women mated to men of their own ABO phenotype. Group AB patients tend to have rapidly progressive tumours which do not respond well to chemotherapy.

While the authors felt that the data indicated how strong were the genetic factors affecting the development of choriocarcinoma, these were extremely difficult to explain on an immunological basis and did not fit in with the hypothesis put forward above. The matter is made still more complicated by a consideration of the HLA system, for here it was found that the patients with trophoblastic neoplasia were not more compatible with their husbands for the HL-A system than a comparable group of normal couples.

The evidence from the 18 families studied indicated that the influences exerted by the ABO and HLA systems in choriocarcinoma are independent of each other. Though there was a suggestion that the chance of survival was greatest among the patients who were most incompatible with their husbands, probably other factors influence prognosis more profoundly than the degree of HLA incompatibility.[36]

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Clinical Problems

Rehabilitation of the Elderly Lower-limb Amputee

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Introduction

About 4,500 new patients are referred each year to the limb-fitting centres of England and Wales after lower-limb amputation and a further 500 are referred after amputation of an upper limb. The prosthetic fitting and subsequent training in most cases are managed on an outpatient basis.[1]

Chilvers and Browse[2] have shown that in order to settle their social problems a high proportion of lower-limb amputees tend to occupy general surgical beds, which are not equipped for the length of stay needed to achieve limb fitting. When the Oxford Artificial Limb and Appliance Centre was opened in September 1968 it was proposed that amputees from the Oxford region whose social and medical background presented problems in prosthetic fitting and usage should be admitted to Mary Marlborough Lodge, the 18-bedded disabled living research unit in the Nuffield Orthopaedic Centre, to which the Artificial Limb and Appliance Centre is physically linked. Thus prosthetic fitting at the appliance centre can be integrated with the comprehensive rehabilitation programme organized in Mary Marlborough Lodge. Since 1968 this proposition has been implemented, and Mary Marlborough Lodge has been admitting about 100 amputees each year.

Patients have been admitted initially for assessment and training at the time of provision of their preliminary pylons and again, if indicated, at the time of issue of their definitive limbs. To evaluate the effect of such inpatient training, to clarify the indication for future admission, and to establish the types of problem being handled we decided to carry out a survey and follow-up on some of the amputee patients who had been admitted to Mary Marlborough Lodge from September 1968 to December 1969 inclusive. The survey was limited to the 50 patients who during this time were admitted to the unit for fitting, issue, and mobility and independence training with primary rocker pylons after lower-limb amputation.

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Patients

During the period under review 149 lower-limb amputees were referred to the appliance centre. Two-thirds of these patients were in the geriatric age group (70% were over 60 years old and 50% were over 70 years old), and nearly three-quarters had their amputation because of vascular insufficiency (Tables I and II).

TABLE I—*Age Distribution of Patients admitted to Oxford Artificial Limb and Appliance Centre during September 1968 to December 1969*

	Age in Years								Total
	<30	30—	40—	50—	60—	70—	⩾80	Not known	
No. of patients	9	3	9	13	32	47	25	11	149

TABLE II—*Reason for Amputation in the 149 Patients*

Vascular insufficiency	82	Chronic infection	5
Vascular insufficiency with diabetes	28	Congenital deficiency	2
Trauma	9	Unknown	15
Malignancy	8		

Altogether 50 (33%) of these patients (36 males and 14 females) were admitted to the disabled living unit during the period under consideration. An analysis of the remaining 99 patients (66%) who were not admitted is given in Table III. Of the 50 patients reviewed 10 were double amputees admitted for the first fitting of the short rocker pylons, after the amputa-